

LETTER

Tree carbon allocation explains forest drought-kill and recovery patterns

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Abstract

The mechanisms governing tree drought mortality and recovery remain a subject of inquiry and active debate given their role in the terrestrial carbon cycle and their concomitant impact on climate change. Counter-intuitively, many trees do not die during the drought itself. Indeed, observations globally have documented that trees often grow for several years after drought before mortality. A combination of meta-analysis and tree physiological models demonstrate that optimal carbon allocation after drought explains observed patterns of delayed tree mortality and provides a predictive recovery framework. Specifically, post-drought, trees attempt to repair water transport tissue and achieve positive carbon balance through regrowing drought-damaged xylem. Furthermore, the number of years of xylem regrowth required to recover function increases with tree size, explaining why drought mortality increases with size. These results indicate that tree resilience to drought-kill may increase in the future, provided that CO₂ fertilisation facilitates more rapid xylem regrowth.

Keywords

Carbon metabolism, CO₂ fertilisation, drought, hydraulic-carbon coupling, lagged mortality, optimality theory, plant hydraulics, stem respiration, vegetation model, xylem damage.

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INTRODUCTION

Observations of drought-driven tree mortality worldwide over the past decade have raised concerns about the future of Earth's forests in a changing climate (Barber *et al.* 2000; van Mantgem *et al.* 2009; Carnicer *et al.* 2011; Anderegg *et al.* 2012b; Reichstein *et al.* 2013; Allen *et al.* 2015; Brien *et al.* 2015; McDowell *et al.* 2018). A continuation or escalation of the observed trends, reflecting widespread tree mortality, could cause a substantial positive feedback, affecting global climate by releasing large amounts of carbon into the atmosphere within a relatively short period (Cox *et al.* 2000), changing the hydrological cycle (Khanna *et al.* 2017), and affecting surface energy budgets (Bonan 2008). Given that live forest biomass represents ~363 Pg C (equivalent to ~170 ppm CO₂ if released to the atmosphere) and intact forests sequester ~2.3 Pg C annually, or approximately 25% of annual anthropogenic carbon emissions (Pan *et al.* 2011), it is critical to understand the mechanisms responsible for drought-induced forest dieback.

There is not yet a widely accepted physiological mechanism that explains all of the observed phenomena associated with forest drought-kill, despite its fundamental importance in forecasting future carbon cycle feedbacks with climate change. During prolonged droughts or after a drought event, trees have been observed to maintain normal diameter growth rates

in some instances (Anderegg *et al.* 2013; Rowland *et al.* 2015; Berdanier & Clark 2016; Herguido *et al.* 2016) while simultaneously reducing leaf and fine root area, only to die in delayed mortality events several years post-drought. However, even with post-drought stem growth, drought-damaged trees show impaired conductivity up until death (Anderegg *et al.* 2012a). In addition, larger trees have been observed to suffer higher rates of drought-induced mortality compared to smaller trees (Bennett *et al.* 2015; Rowland *et al.* 2015), even though larger trees generally have bigger root systems that can access deep water reservoirs (Chitra-Tarak *et al.* 2018). Current leading hypotheses identify an interaction between hydraulic failure and plant carbon metabolism as the principal mechanism underlying drought-induced tree mortality (McDowell *et al.* 2008; Sala *et al.* 2012; Sevanto *et al.* 2014; Savage *et al.* 2016). Yet, when placed in the context of the drought-kill observations, these hypotheses do not explain why drought-induced hydraulic failure of the xylem or carbon starvation result in (1) multi-year post-drought deteriorations preceding mortality and (2) increased mortality in larger trees.

Here, we used a combination of meta-analysis and tree physiological models based on optimality theory to ask: (1) Can xylem damage result in lagged tree mortality? (2) Is there a xylem damage threshold below which trees will eventually starve due to a net negative carbon balance? If so, how do environmental conditions influence the xylem damage

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threshold? (3) Can xylem damage explain why large trees suffer higher mortality rates compared to smaller trees post-drought?

MATERIALS AND METHODS

Overview

We first performed a meta-analysis of existing drought-induced tree mortality studies to quantify the multi-year lags in tree mortality post-drought (Fig. 1, Table S1). We next tested how hydraulic damage during drought affects post-drought carbon gain and could lead to delayed mortality by building a model of a tree consistent with existing physiological knowledge. Importantly, the tree model coupled carbon allocation to local environmental conditions through gas exchange and plant hydraulic transport. We imposed a drought event that permanently damaged a fraction of the tree's xylem, representing the process of non-reversible xylem embolism whereby tension in the tree's water transport tissue results in air bubble formation, rendering the xylem unusable. After the drought event, the tree was subjected to normal growing conditions. We assumed that tree carbon allocation evolved to maximize instantaneous net primary productivity (photosynthesis minus respiratory costs) as a proxy for plant fitness (Franklin *et al.* 2012), and solved for the optimal post-drought allocation of carbon among xylem, leaves and fine roots. We analysed model predictions for optimally-allocating trees to determine how post-drought tree growth and mortality rates depended on the extent of xylem damage, tree size and post-drought environmental conditions. We then compared these results to observed trends in drought-induced tree mortality.

Photosynthesis model

The photosynthesis model calculated net whole-plant photosynthesis dependent on the following input environmental conditions: atmospheric vapour pressure deficit (VPD), soil water potential and atmospheric CO₂ concentration. Tree biological parameters that influenced whole-plant photosynthesis included functional tree xylem area, leaf area, tree size, as well as a number of physiological traits (Table S2). The model assumed that photosynthesis was not light limited during daytime (comprising 50% of a given day). All leaves experienced the same VPD and CO₂ and all fine roots experienced the same soil water potential. A full description of the photosynthesis model and model code is available as Supporting Information.

Drought recovery model

The allocation strategies of photosynthetic carbon and accessible non-structural carbohydrate reserves (NSC) after drought events that caused non-reversible xylem damage were derived using optimality theory whereby trees maximized instantaneous carbon gain (Franklin *et al.* 2012). For the purposes of this study, we defined NSC as starch NSC, not soluble sugars required for osmoregulation (Martínez-Vilalta *et al.* 2016).

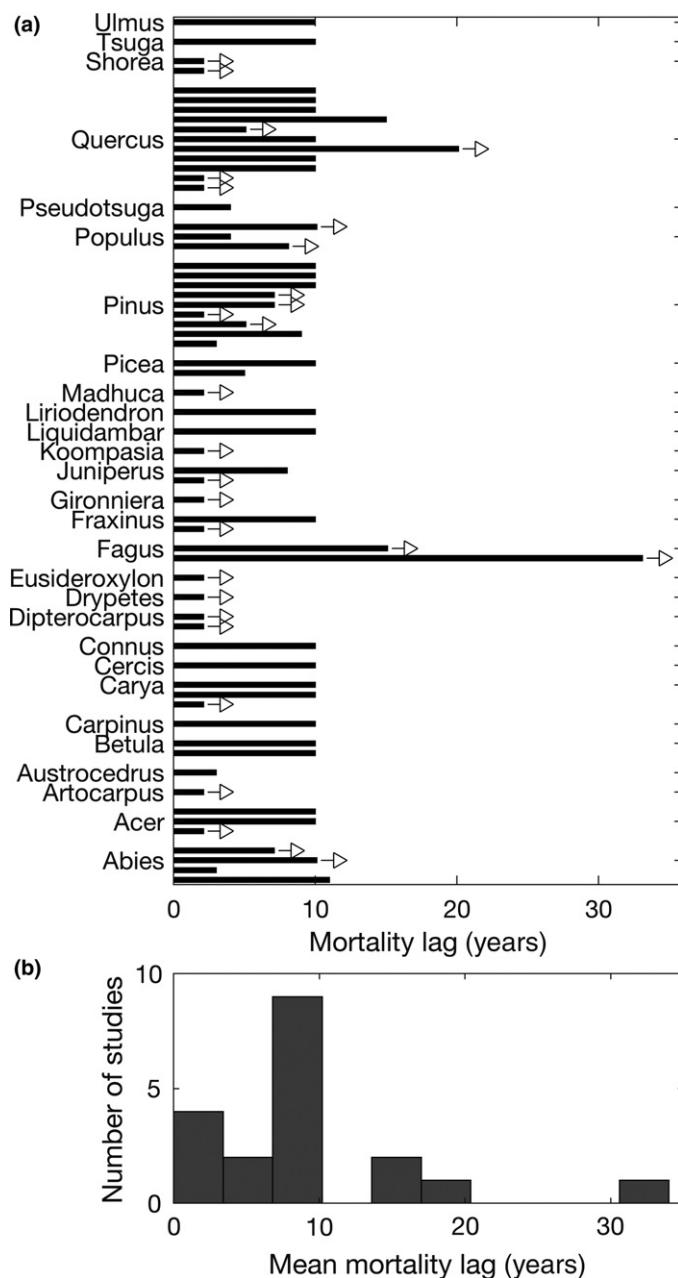


Figure 1 Delayed mortality following drought is a phenomenon that has been documented in a variety of tree genera globally. (a) Mortality lag in years for 28 tree genera. Black bars indicate lag for different studies. Arrows indicate if elevated mortality was ongoing when the study concluded. (b) Histogram of the mean mortality lag across all species for a given study documented at the time of study termination. See Table S1 for details on individual studies including species and location.

During the recovery process, trees were exposed to a constant environment with fixed VPD, soil moisture and atmospheric CO₂. We assumed that the growing season comprised 40% of the year (consistent with many temperate ecosystems) and considered the following system of ordinary differential equations:

$$\frac{dC}{dt} = A_n - W \quad (1a)$$

$$\frac{dX}{dt} = Wu - m_X X \quad (1b)$$

$$\frac{dL}{dt} = W(1 - u) - m_L L \quad (1c)$$

In eqns. (1a–c), C is the NSC, t is the time in months, A_n is whole-plant net photosynthesis, W is the sucrose loading rate from the storage to the phloem, X is the xylem biomass, L is the leaf and fine root biomass, which are linearly related through a constant (Table S2), and m_X and m_L are the turnover rates of the xylem and leaves/fine roots respectively. $u \in [0, 1]$ is the variable optimised in this system, and represents the fraction of translocatable C invested in xylem reconstruction. A_n includes both carbon gains due to carbon fixation and respiration losses resulting from leaf/fine root respiration, growth respiration, xylem respiration and cambium/phloem respiration. The phloem loading rate, W , is dependent on NSC concentration and tree size (because total plant biomass per time step increases with tree size), is independent of xylem pressure, and assumed to obey Michaelis-Menten kinetics (De Schepper & Steppe 2010) (Table S2). We confirmed that our assumption that leaf and root biomass vary jointly was reasonable (given a uniform soil water potential) by optimising leaf area and root area separately, which showed that both scale linearly with xylem (Fig. S1). We assumed no phloem turnover or damage during drought and no height growth. Thus, our results should be interpreted as optimistic recovery estimates. This system (eqn. (1a–c)) is at equilibrium (whole-plant net carbon balance of zero) when net whole-plant photosynthesis exactly covers leaf and xylem turnover costs ($A_n = m_X X + m_L L$).

In our model, the leaf area that Maximizes photosynthesis (i.e. $\frac{\partial A_n}{\partial L_a} = 0$, where A_n is defined in the Supplemental Methods eqn. (S-23) and L_a is leaf area) scales linearly with functional xylem area as:

$$L_{a,opt} \propto X_a \quad (2)$$

where $L_{a,opt}$ is the optimal leaf area for a given functional xylem area X_a (see the maximums in Fig. 2a). Leaf and xylem area are linearly related to leaf and xylem biomass, so it is also true that optimal total leaf biomass (L_{opt}) scales with xylem biomass

$$X(t) = hL_{opt}(t) \quad (3)$$

where h is a constant for any given tree height (Fig. S2). Thus, xylem biomass growth also scales linearly with total biomass growth

$$\frac{dX(t)}{dt} = h \frac{dL(t)}{dt} \quad (4)$$

Substituting eqns. (1a–c) and (3) into eqn. (4)

$$u(t) = \frac{X(t)}{X(t) + L_{opt}(t)} \frac{W(t) - (m_L + m_X)L_{opt}(t)}{W(t)} \quad (5)$$

which defines the optimal allocation strategy to xylem during the recovery period.

We assumed that the tree began the drought recovery period with non-reversible xylem damage and was deficient in functional xylem biomass relative to a healthy tree allometry. Given that leaf biomass is more flexible than structural woody biomass in trees, and that leaf abscission has been observed

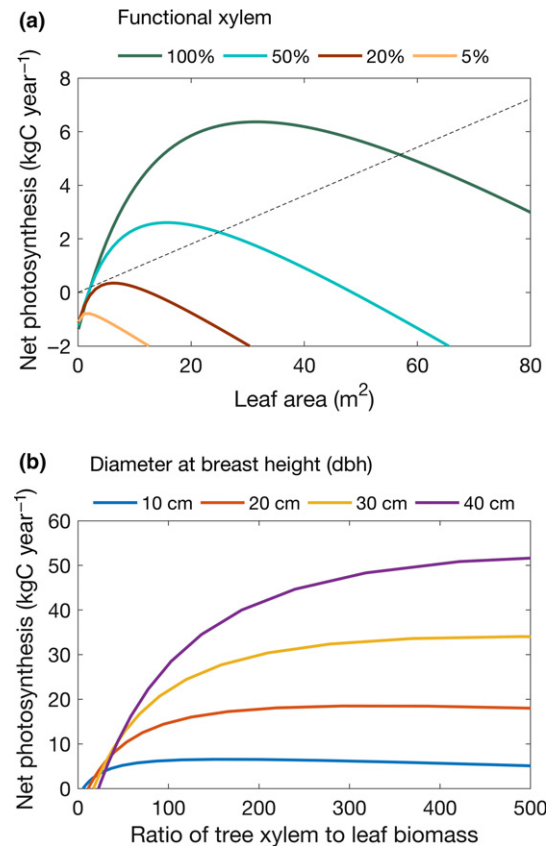


Figure 2 At a given tree size, whole-plant photosynthesis is an integrated function of both leaf and xylem tissue, however, the ratio of xylem biomass to leaf biomass that maximizes photosynthesis increases with tree size. (a) Net photosynthesis for a tree of size 20 cm diameter at breast height (dbh) as a function of tree leaf area for differing amounts of functional xylem ranging from 5 to 100% (coloured lines). Dashed line indicates the carbon cost of leaf turnover, below which trees are in net negative carbon balance. (b) Whole-plant photosynthesis for trees of different size classes ranging from 10 to 40 cm dbh (coloured lines) as a function of the ratio of xylem to leaf biomass.

during and post-drought in response to severe water limitation (Carnicer *et al.* 2011), we allowed drought-damaged trees to immediately abscise excess leaves to maximize instantaneous carbon gain at their given level of functional xylem. We also assumed that NSC stores contained enough carbon to replace the fine roots and leaves for a tree with a healthy allometry twice over. The system of eqns. (1a–c) was then solved numerically until xylem and leaf area were fully recovered or the tree died due to starvation in the case that accessible NSC was depleted to less than 10% of its initial value.

Under the above defined drought recovery framework, carbon biomass trajectories and $u(t)$ were dependent on tree size and time (Fig. S3). Using this recovery framework, we examined whole-plant photosynthesis as a function of tree leaf area, functional xylem and environmental conditions for both optimally allocating trees and tree recovering under non-optimal conditions. Finally, we defined the 'xylem damage threshold' (the xylem damage below which a tree is negative carbon balance and cannot recover) and looked at its sensitivity to soil water, CO_2 and VPD.

Literature search of observed post-drought mortality

We compiled drought mortality data drawing from two meta-analyses of drought-induced tree mortality (Anderegg *et al.* 2016; Cailleret *et al.* 2016) and a Google Scholar search of studies documenting drought-induced tree mortality. Studies that satisfied the following constraints were included: (1) Mortality was attributed to drought stress; (2) no other major disturbance (e.g. insects, pathogens, fire or harvest) had occurred that could drive mortality; (3) mortality time period was documented; and (4) measurements continued after the drought event terminated so that elevated mortality post-drought could be documented if present. This led to the identification of 20 published studies spanning 28 genera and 16 distinct locations around the globe (Fig. 1 and Table S1).

In our sample of studies, we used the most recent single specific drought event to calculate the mortality time lag after drought disturbance. Although in some studies, mortality may have resulted from declines due to compounding drought events, our analysis was intended to demonstrate that multi-year lagged mortality post-drought is a widespread phenomenon. Thus, our estimates can be viewed as a lower bound for the time that a tree can continue to grow after incurring terminal drought damage. Furthermore, although all included studies continued measurements after the drought event terminated, not all studies continued long enough to document a return in mortality rate to background levels. We indicate these studies as a lower bound estimate in our results (Fig. 1a and Table S1). We looked at mortality lag for individual genera (Fig. 1a), and mean mortality lag across all species for each individual study (Fig. 1b) because study termination time and design substantially impacted estimated mortality lag. Overall, substantial lags in mortality, up to 30 years post-drought, were documented globally across studies, demonstrating that multi-year lagged mortality post-drought is a widespread phenomenon.

Modelled post-drought tree diameter increment

We calculated the post-drought diameter increment for optimally allocating trees with a range of severities of xylem damage, including trees that were unable to regain positive carbon balance and thus depleted their NSC reserves, and trees that were able to repair damage post-drought. We compared predicted diameter increments to observed normalised ring widths of branches of aspen trees that survived and suffered mortality post-drought (where normalised ring width is ring width normalised by the total xylem diameter for each individual branch) (Anderegg *et al.* 2013). We calculated predicted time to mortality, defined in this study as time to NSC reserve depletion, assuming that a growing season (1 year of growth) comprised of 4 months of the year (roughly May–August) based on the location of the aspen measurements. We also examined the sensitivity of post-drought diameter increment for trees with terminal hydraulic damage to different maximum sucrose loading rates (Fig. S4). Depending on sucrose loading rate from NSC to repair damaged tissues, optimal carbon allocation can explained the range of observed trends prior to mortality documented in our meta-analysis

(Table S1), including normal to decreased stem growth rates and extended multi-year lags concurrent with tree stem growth preceding mortality.

Model sensitivity tests

We performed extensive sensitivity analyses to examine the sensitivity of optimal tree biomass allocation to leaves, fine roots and xylem to variations in key environmental and eco-physiological parameter choices including variations in specific leaf area, V_{cmax} , xylem and phloem/cambium respiration, plant hydraulic conductivity, biomass allocation to leaves relative to fine roots (q), VPD and atmospheric CO_2 . Biomass allocation to leaves, fine roots and xylem showed qualitatively identical trends with tree size independent of choice for plant traits and environmental conditions. Model sensitivities for key traits and environmental conditions are shown in Fig. S5.

Model limitations

In the current model formulation and model predictions, we neglected the effects that competition, shading, variability in the tree size-to-height relationship under differing environmental conditions, variability in meteorology, and temperature have on the recovery time. An analysis of the impacts of different stomatal optimised behaviour hypotheses could also provide insight and species/trait-specific observational comparisons for how changing water stress impacts tree hydraulic-carbon coupling (Manzoni *et al.* 2011; Anderegg *et al.* 2018). Although we do not account for stochasticity and variability in meteorology in our model optimization, our recovery framework is broadly applicable and can inform process-based drought recovery formulations in ecosystem models. Furthermore, model predictions for drought recovery are consistent with observed spatial trends in recovery time and canopy tree size and we contend that they will help inform understanding of how atmospheric VPD and CO_2 fertilisation will impact the severity of droughts on tree mortality and carbon drawdown.

RESULTS

In our model, trees reached and then maintained an optimal ratio of xylem to leaf and fine root biomass. This strategy maximized instantaneous whole-plant carbon gain by balancing the benefits of increased leaf area with the costs of respiration and water stress under a given set of environmental conditions (Fig. 2a; Fig. S6). An over-allocation to leaf biomass relative to xylem increased plant water stress, triggering stomatal closure to prevent cavitation, thus decreasing photosynthesis per unit leaf area and increasing leaf respiratory costs without a compensating increase in productivity. Conversely, under-allocating to leaf biomass relative to xylem increased stem respiration per unit leaf area, thereby decreasing net plant productivity (Fig. S6).

The model showed that, although large trees support more leaves than small trees, the optimal ratio of xylem to leaf and fine root biomass increases rapidly with tree size (Fig. 2b). This trend in xylem to leaf and fine root biomass occurs

because xylem cross-sectional area (S_A) must grow faster than leaf and fine root area, as resistance to water transport (r) is proportional to tree height (H) (i.e. $r \sim H/S_A$). Such a difference in allocation strategy with tree size has been documented in a number of studies (Albrektson 1984; Thompson 1989; van Hees & Bartelink 1993; Shelburne *et al.* 1993; Vanninen *et al.* 1996; Magnani *et al.* 2000). Although, it should be noted that this trend is not ubiquitous in the observations, likely due to co-occurring changes in xylem anatomy and other hydraulic characteristics throughout a tree's lifetime (Magnani *et al.* 2000). However, barring substantial anatomical adaptation, larger, taller trees are less efficient at supplying water for transpiration. As a result, the number of years of growth required to build the xylem to support a tree's canopy increases with tree size. These scaling arguments are confirmed by allometric and tree growth data (Farrior *et al.* 2013), and are qualitatively robust to variations in environmental and ecophysiological model parameter choices (Fig. S5).

After a tree has suffered drought-induced xylem damage, the model-predicted optimal recovery strategy required shedding of excess leaves and fine roots, as is widely observed (Carnicer *et al.* 2011), so as to avoid the increased water stress associated with over-extending tree leaf area relative to xylem area (Fig. 2a; Fig. S6). Leaf and fine root pruning was followed by persistent stem growth post-drought as a recovering tree allocated photosynthetic gains and accessible NSCs to rebuild the damaged xylem, leaves and roots in proportion to maximize instantaneous carbon gain. Nonetheless, trees that suffered severe xylem loss were still net negative in carbon gain because of the respiration costs of the cambium and phloem (Fig. S7). These cambium and phloem respiratory costs can comprise the majority of tree stem respiratory burden (Stockfors & Linder 1998), particularly in larger trees, making it impossible for the trees to recover from severe xylem damage.

Optimal carbon allocation to damaged xylem explained the widely observed multi-year lags in mortality post-drought (Table S1; Fig. 1). Our optimal allocation model predicted that trees maintained stem growth rates to replace non-recoverable embolised xylem (Rowland *et al.* 2015) because whole-plant carbon gain was coupled to the plant's hydraulic transport system. Thus, rapid repair of a tree's hydraulic transport system was the only pathway to obtain a net positive carbon balance. However, some trees ultimately succumbed to a depletion of accessible NSC reserves that lead to tissue carbon starvation and mortality during the repair process. The time lag between terminal drought damage to xylem and ultimate NSC depletion and mortality depended on the amount of xylem damage, the size of the pool of NSC reserves available for metabolism, and growing season length. For realistic parameter values and a growing season comprising 40% of the year, this time lag could extend several years (Fig. 3a), as observed in empirical studies (Fig. 3b and see Fig. 1, Fig. S4, Table S1). Naturally, the maximum survivable xylem damage from which a tree could recover increased both with available water and the atmospheric CO_2 concentration (due to the CO_2 fertilization effect on photosynthesis) (Fig. 4a–b), a finding which complements global observations that CO_2 fertilisation decreases recovery time post-drought (Schwalm *et al.* 2017).

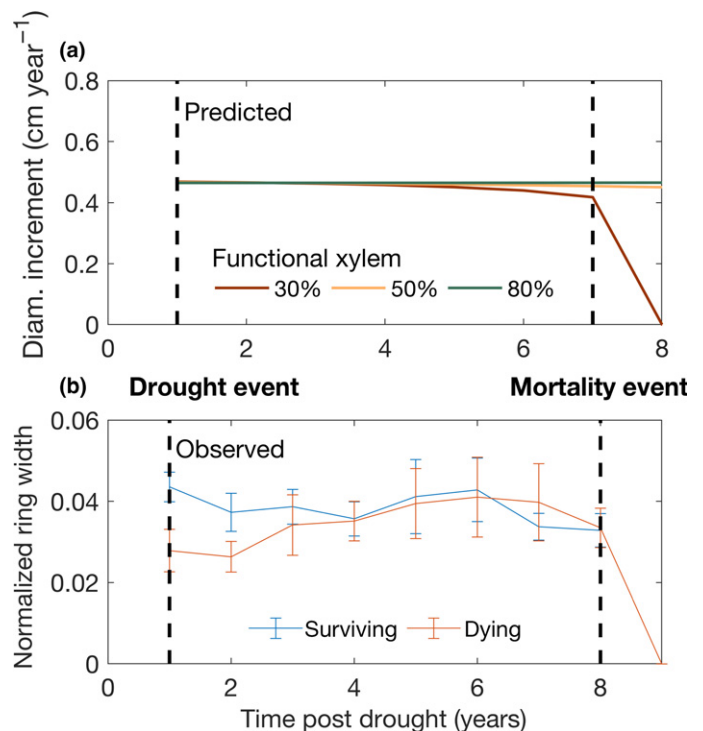


Figure 3 Optimal carbon allocation explains observed lags in mortality in drought-damaged trees. (a) Predicted post-drought diameter increment for trees with different levels of hydraulic damage (as percent functional xylem). Mortality occurs when diameter increment declines to zero. (b) Observed normalised ring width for aspen tree branches located in southwestern Colorado, USA that survive and die in lagged mortality events post-drought (Anderegg *et al.* 2013). See Methods for details and Table S1, Fig. 1 for other documented lagged mortality events post-drought.

Optimal carbon allocation to damaged xylem also predicted that larger trees are more vulnerable to drought damage relative to smaller trees (Fig. 4c). First, the maximum survivable xylem damage decreased as tree size increased, both because phloem and cambium respiration increase with tree size and because frictional and gravitational resistance to water flow increase with size. Moreover, large trees with survivable xylem damage took longer to recover than small trees (Fig. 4c), because the optimal ratio of xylem to leaf biomass increase with tree size (Fig. 2b). Finally, although not in the model, older xylem, which is present to a greater extent in larger trees, may be less resistant to embolism at less negative water potentials due to mechanical stressing of xylem tissue (Hacke *et al.* 2001). Such physiological responses support findings that larger trees may experience mortality more frequently for a given level of soil moisture or atmospheric water stress.

Furthermore, even if trees allocated carbon optimally for the climates in which they had evolved, climate conditions could be suboptimal during any one point in time due to weather variability and/or changes in mean climate (Jump *et al.* 2017). As a result, model-predicted recovery times and recovery ability should be viewed as optimistic. Trees recovering under drier-than-normal conditions over-extend leaf area relative to xylem area, which resulted in decreased whole-plant carbon gain (Fig. 2a) and decreased the minimum initial xylem damage that caused mortality (Fig. 4d). Conversely, trees recovering under wetter-than-

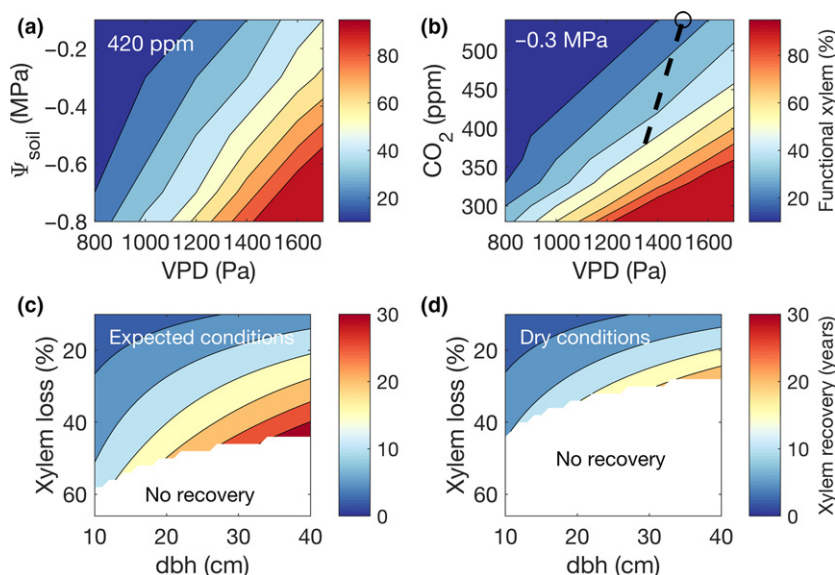


Figure 4 The hydraulic damage extent below which a tree cannot recover net positive carbon balance is strongly influenced by environmental conditions, tree size and optimal carbon allocation. (a–b) Sensitivity of xylem damage threshold (indicated by colour shading) to soil water (Ψ_{soil}), atmospheric vapour pressure deficit (VPD) and atmospheric CO_2 . Black dashed line tracks the projected changes in CO_2 :VPD from circa 2000 to 2050 (denoted by the black open circle) according to the steepest emissions scenario based on warm-season VPD estimates (Williams *et al.* 2012). (c–d) Sensitivity of tree recovery time (indicated by colour shading) to xylem loss and tree size. White shading denotes damage space where trees cannot recover. Panel (c) shows time to recovery for trees able to allocate carbon optimally for realised environmental conditions. Panel (d) shows time to recovery for trees over allocating to leaves due to an unexpected 10% increase in VPD during recovery.

expected conditions recovered more rapidly due to decreased water stress, despite under-allocating carbon to leaf biomass.

Model trends were robust regardless of variations in key physiological plant traits and environmental parameter choices (Fig. S5). Specifically, the emergent allometric constraint from optimal tree biomass allocation to leaves, fine roots and xylem was qualitatively identical with tree size, independent of trait parameter choice and environmental conditions. This was true for variations in critical photosynthetic parameters, stem respiration rates, the allocation of carbon to leaves relative to roots, hydraulic parameters, as well as for variations in water availability and CO_2 .

DISCUSSION

This study documents for the first time that delayed mortality postdrought is a global phenomenon (Fig. 1, Table S1) and demonstrates that optimal carbon allocation and metabolism explain widespread and unresolved patterns associated with tree drought mortality. Specifically, we find that observed multi-year lags in mortality post-drought can be attributed to slow carbon starvation while trees attempt to rebuild damaged xylem to increase whole-plant carbon gain (Fig. 2a). Second, increased tree size decreases the probability of recovering from drought-induced hydraulic damage because large trees must: (1) regrow more years of lost xylem to recover, substantially increasing recovery time and (2) balance higher stem respiration costs related to maintaining a larger cambium and phloem. Third, increased productivity resulting from CO_2 fertilisation may enable more rapid recovery of drought-damaged trees in the future, increasing drought survival independent of increases in water use efficiency.

Furthermore, several lines of ecological evidence link optimal carbon allocation after drought to observed phenomena related to post-drought tree growth and response to insect attack. First, optimal carbon allocation can explain observations of robust stem growth preceding mortality in drought-damaged trees (Anderegg *et al.* 2013; Rowland *et al.* 2015; Berdanier & Clark 2016; Herguido *et al.* 2016) and why pre-mortality growth is higher in drought-killed trees (even after the inciting drought) than in trees that suffer mortality due to other forms of tissue carbon starvation such as competition (Cailleret *et al.* 2016). In the case of drought-damaged trees, it is necessary to regrow xylem tissue post drought to regain hydraulic function and photosynthetic capacity. In contrast, increased radial xylem (stem) growth does not benefit trees suffering carbon starvation from other causes such as light limitation.

Second, long-term hydraulic-driven starvation may also result in drought-damaged trees strategically minimising non-essential carbon costs and diverting resources to growth to decrease recovery time and increase recovery probability. For example, investment in tree herbivory defences may be reduced as a result of carbon stress during recovery. This would explain data suggesting increased vulnerability to pests after a drought (Hicke *et al.* 2012; Allen *et al.* 2015; Gaylord *et al.* 2015), and increased vulnerability of large trees (Pfeifer *et al.* 2011), which are subjected to substantially longer recovery periods (and thus experience a longer time during which reservoirs of herbivore deterrents remain depleted) compared to small trees.

Similarly, if drought damage is so severe that death is inevitable in the average post-drought environment, then a tree could over-allocate to leaves, hoping for a string of wet years and the small chance that an over-allocation to leaves would increase net whole-plant carbon gain enough to put the tree

into positive carbon balance. However, under normal conditions, the overextension of tree canopy area might result in increased tension in the xylem, impaired conductivity (Anderegg *et al.* 2012a) and a net decrease in whole-plant carbon gain. In general, such allocational gambling allows proximate mechanisms to be easily confounded with the ultimate cause of tree mortality. That is, observations would seem to point to many causes of drought-related mortality and little parsimony, when in fact all would have the same underlying cause associated with plant metabolism and allocation after drought to restore hydraulic function.

Finally, if long-term hydraulic-driven starvation is indeed the underlying cause for drought-related mortality, mortality following a drought event could be expected to follow a bimodal distribution with a strong mortality peak during the drought event and a secondary peak after several growing seasons (Fig. 5). The primary peak is diagnostic of severe drought damage resulting in immediate mortality in the majority of trees. However, trees that do not die during the drought but are terminally damaged (i.e. trees that cannot reach an allometry that would allow them to achieve positive carbon gain long-term), might survive for many years in the attempt to recover xylem functionality. Depending on available NSC concentration and maximum sucrose loading rate from NSC to repair damaged tissues, optimal carbon allocation can explain the range of observed tree growth trends prior to mortality including normal to decreased stem growth rates preceding mortality (Fig. S4; Table S1). Currently, data availability, differences in study methods and confounding factors such as compounding drought events make it difficult to isolate the hypothesized bimodal signal from observations of lagged drought mortality, but model predictions provide a targeted framework for future observational studies and support current evidence related to post-drought growth and mortality (Fig. 1, Table S1).

Multiple studies have reported that trees suffering delayed mortality during or post-drought still have remaining NSC at the time of mortality (Anderegg *et al.* 2012a; Rowland *et al.* 2015), which seems at odds with carbon starvation. Although a recent meta-analysis of physiological data associated with drought-induced tree mortality documented NSC reduction in 63% of boreal and temperate angiosperm species, a similar decrease in NSC was not observed in drought-killed seedlings of tropical angiosperm species (Adams *et al.* 2017). Thus, declines in NSC with drought mortality can be observed, but are not ubiquitous (Anderegg *et al.* 2012a; Rowland *et al.* 2015; Adams *et al.* 2017). However, a fairly high level of remaining NSC may be expected in drought-killed trees, given the role of NSC in maintaining hydraulic function such as osmoregulation (Sala *et al.* 2012; Martínez-Vilalta *et al.* 2016), and that damaged xylem leads to turgor loss in the phloem which limits or even prevents access to NSC reserves (Sala *et al.* 2010; Sevanto *et al.* 2014). Our model attribution of tree drought mortality to the combined loss of plant hydraulic function coupled with carbon metabolic processes is in line with these observations.

These results also have important implications for future tree drought resilience in a changing climate. In particular, we show that atmospheric CO₂ fertilization has the potential to reduce drought-kill caused by projected increases in VPD

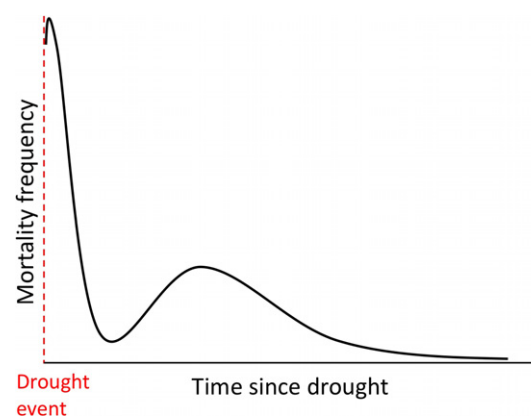


Figure 5 Schematic of expected mortality frequency after a drought event (indicated by dotted red line) explained by the optimal carbon allocation hypothesis from this study.

under anticipated warming trends (Fig. 4b). Such results are consistent with observations that CO₂ fertilization acts to decrease productivity recovery time post-drought (Schwalm *et al.* 2017), and other modelling studies that project a decrease in drought-driven mortality and enhance productivity with climate change (Lloyd & Farquhar 2008; Liu *et al.* 2017). However, caution should be used when interpreting these predictions, because they omit effects of climate variability and ecophysiological trait adaptation (Drake *et al.* 1997). Furthermore, the interaction between tree carbon allocation strategy (Fig. 4d) and climate change will also influence future forest drought resilience.

By assuming an optimal approach to carbon allocation post-drought as a proxy for tree fitness, this study provides the foundation to link current trait-based statistical analyses into a mechanistic framework that can be used to better predict (1) how trees may respond to drought under future climate conditions, and (2) to target future measurements for rigorous validation of our proposed mortality mechanism. Furthermore, a currently major shortcoming of most vegetation models is the lack of mechanistic tree hydraulic and mortality processes (Sitch *et al.* 2008; Trugman *et al.* 2018), which can cause large uncertainty in estimates of the terrestrial carbon sink (Friend *et al.* 2014; Trugman *et al.* 2018). These results highlight that coupling carbon allocation to environmental conditions through plant hydraulics is a critical mechanism governing drought recovery and mortality. Thus, dynamic carbon allocation schemes and vegetation hydraulics are necessary processes that should be incorporated in the next generation of vegetation models to accurately project how changes in mean climate, climate variability and ecophysiological trait plasticity may interact with drought-kill and feedback on the terrestrial carbon cycle.

AUTHORSHIP

ATT, MD, MKB and SWP designed the study with input from DM and WRLA. ATT, MD and MKB built the model. CS and BS contributed data and analytical tools. ATT performed the analysis and wrote the paper, with all authors contributing comments.

DATA ACCESSIBILITY STATEMENT

Should the manuscript be accepted, the data supporting the results will be archived on the Hive, the University of Utah's Open Access Institutional Data Repository and the data DOI is 10.7278/S5N29V4F.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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